WHAT IS CLAIMED IS:

1. A non-murine heart valve cell containing an exogenous nucleic acid that encodes a polypeptide having nitric oxide synthase activity.

2. The cell of claim 1, wherein said cell is an endothelial cell.

3. The cell of claim 1, wherein said cell is a myocyte.

4. The cell of claim 1, wherein said polypeptide is endothelial nitric oxide synthase.

5. An isolated heart valve cusp, wherein a cell of said cusp contains an exogenous nucleic acid that encodes a polypeptide having nitric oxide synthase activity.

6. The cusp of claim 5, wherein said cell is porcine.

7. The cusp of claim 5, wherein said cell is human.

8. The cusp of claim 5, wherein said polypeptide is endothelial nitric oxide synthase.

9. A method for making a bioprosthetic heart valve, said method comprising:

a) obtaining a heart valve cusp, and

b) introducing nucleic acid into a cell of said cusp, wherein said nucleic acid encodes a polypeptide having nitric oxide synthase activity.

10. The method of claim 9, wherein said cell is porcine.

11. The method of claim 9, wherein said cell is human.

12. The method of claim 9, wherein said polypeptide is endothelial nitric oxide synthase.

13. The method of claim 9, wherein said nucleic acid is introduced into said cell via adenoviral-mediated nucleic acid transfer.

14. The method of claim 9, wherein said nucleic acid integrates into the genome of said cell.

15. The method of claim 9, wherein said method comprises fixing said cusp.

16. The method of claim 15, wherein said fixation step occurs after said introduction step.

17. The method of claim 9, wherein said method comprises freezing said cusp.

18. The method of claim 17, wherein said freezing step occurs after said introduction step.

19. A method for slowing the degeneration of a heart valve within a non-murine mammal, said method comprising introducing nucleic acid encoding a polypeptide having nitric oxide synthase activity into a cell of said heart valve such that said polypeptide is expressed.

20. The method of claim 19, wherein said introduction step is performed *in vitro*.

21. The method of claim 19, wherein said heart valve is an autograft.

22. The method of claim 19, wherein said heart valve is an allograft.

23. The method of claim 19, wherein said heart valve is a xenograft.

24. The method of claim 19, wherein said method comprises administering an inhibitor of

hydroxymethylglutaryl coA reductase activity to said mammal.

25. The method of claim 24, wherein said inhibitor comprises pravastatin, atorvastatin, simvastatin, or lovastatin.

26. A method for slowing heart valve degeneration, said method comprising:

a) identifying a mammal at risk of developing heart valve degeneration, and

b) administering an inhibitor of hydroxymethylglutaryl coA reductase activity to said mammal.

27. The method of claim 26, wherein said mammal contains a heart valve replacement.

28. The method of claim 26, wherein said mammal has congenital valvular disease.

29. The method of claim 26, wherein said mammal has bicuspid valvular disease.

30. A method for treating carcinoid heart disease in a mammal, said method comprising administering a serotonin receptor antagonist to said mammal.

31. The method of claim 30, wherein said antagonist is specific for a $5HT_{1B}$ receptor.

32. The method of claim 30, wherein said antagonist comprises a β-blocker.

33. The method of claim 30, wherein said antagonist is pindolol.

34. A method for identifying an inhibitor of heart valve degeneration, said method comprising:

a) contacting heart valve cells with a stimulant such that said cells proliferate,

b) contacting said cells with a test compound, and

c) determining if said test compound reduced the proliferation of said cells, wherein the reduction of proliferation indicates that said test compound is an inhibitor of heart valve degeneration.

35. A method for determining the safety of a drug, said method comprising:

a) contacting heart valve cells with said drug, and

b) determining if said drug induced proliferation of said cells, wherein the induction of proliferation indicates that said drug promotes heart valve degeneration.